FOREIGN-BODY PSEUDO-TUBERCULOSIS OF THE PERITONEUM.

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PSEUDO-TUBERCULOSIS, a term coined by Eberth in 1885, has widened in its application so as to include under its designation all conditions which anatomically resemble genuine tuberculosis, whatever their etiology.

Ophüls, in a valuable survey of this subject in the Reference Handbook of Medical Sciences, Vol. VI, page 778, adopts the following etiological classification:

1. Pseudo-tuberculosis caused by the bacillus Pseudo-tuberculosis Rodentium. Two not altogether convincing instances of human infection are reported in this connection.

2. Pseudo-tuberculosis caused by a bacillus resembling the bacillus of Pseudo-tuberculosis Rodentium. Du Cazal reports two such cases, in one of which nodules the size of a hazelnut were found on the surface of the peritoneum, in the pancreas and liver. Wrede records another instance of such infection in which miliary nodules were found on the peritoneum, as well as in other localities. The causal bacillus possessed acid fast staining properties.

3. Pseudo-tuberculosis caused by other bacilli, as, *e.g.*, bacilli resembling the Diphtheria bacillus.

4. Pseudo-tuberculosis caused by filamentous bacteria. Eppinger records a case of brain abscess. Flexner, under the designation of Pseudo-tuberculosis Hominis, reports an instance in which tubercular-looking lesions were found in the lungs and tubercle-like nodules in the peritoneum. A streptothrix was apparently the pathogenic germ.

5. Pseudo-tuberculosis caused by mould fungi, *e.g.*, pigeons' pseudo-tuberculosis due to the aspergillus mould. Several instances of human mould infection are on record.

6. Pseudo-tuberculosis produced by the organism of Blastomycetes Dermatitis.

7. Pseudo-tuberculosis produced by the fungus coccidiodes. Here the combination of the miliary abscess and miliary tubercles may be very bewildering, as Ophüls has pointed out.

8. Pseudo-tuberculosis caused by animal parasites and their eggs.

9. Pseudo-tuberculosis produced by dead foreign bodies.

The first seven varieties are mainly of interest to the physician, and are becoming clinically more important, since the list of reported cases of lung disease is constantly lengthening, in which symptoms and physical signs suggestive of tuberculosis may occur, together with acid fast bacilli in the sputum, and yet the ailment be non-tubercular in origin. Some of these cases, however, are only clinically pseudo-tubercular, the underlying pathologic lesion not resembling genuine tuberculosis. From what has been said, it will be seen that if the peritoneum be involved in these varieties, it occurs in association with similar lesions elsewhere, and serious errors in intra-abdominal operative work are not apt to occur. The last two varieties, however, possess a special significance for the surgeon, as the following cases will show:

Helbing, in the *Berl. klin. Wochenschr.* 1899, page 714, records as follows: A laparotomy was performed for perityphlitis. Tubercles were seen on the omentum and cæcum. A piece of omentum was removed. With the endeavor to demonstrate the tubercle bacillus, sections were made and suitably stained. In place of the bacillus, the eggs of the tænia worm were found occupying the center of the nodules lying among the giant cells. Helbing ascribes the lesions to the escape of these eggs from a perforated appendix, they acting as chronic localized peritoneal irritants. He credits Miura, of Tokio, with a similar observation, but omits the reference.

Carl Meyer, in the *Beitr. z. Pathol. Anat. u. Allegem. Pathol.* xiii, 1, 1903, reports a case of a woman who had had an abdominal tumor for some years. A few weeks previous to her entrance to the hospital it had evidently ruptured. There was now free fluid in the peritoneal sac. Examination of the aspirated fluid showed cholesterin crystals and red blood cells. Laparotomy demonstrated an ovarian cyst, many peritoneal miliary tubercles, and in their neighborhood fibrinous masses. Macroscopic examination of tissue removed resulted in the diagnosis of a dermoid ovarian cyst and tuberculer peritonitis. Microscopic investigation, however, revealed the true nature of these little bodies; they were pseudo-tubercles. In their center were giant cells, and amongst the giant cells, and in some instances enclosed in these cells, were cholesterin crystals. The diagnosis was then changed and naturally—an all important point for the patient—the prognosis. Meyer then refers to several instances of pseudo-tuberculosis of the conjunctiva and iris due to the irritation and enclosure of little hairs, and to experiments showing the formation of giant cells around wound ligatures. He records two other extremely interesting cases.

The first (Von Recklinghausen) concerns a woman who died from pneumonia several months after being laparotomized. A disseminated tuberculosis of the abdomen was found. Microscopic examination of the tubercles, however, showed that it was another instance of foreign-body pseudo-tuberculosis, little pieces of sponge left behind in the abdomen at the time of the operation being found within the nodules.

In the second case, first reported by Hanau in the *Correspondensblatt für Schweizer Aerzte Jahrbuch*, xxi, a man who had had typical symptoms suggestive of gastric ulceration for upwards of five years, was seized twelve days previous to his death with epigastric pain, accompanied by fever. Postmortem revealed a large ulcer saddling the lesser curvature and this had perforated; in the adhesions round about and in the neighboring peritoneum were numerous miliary tubercles. Though the ulcer looked to be of a simple nature, it was suspected to be carcinomatous, these little nodules being regarded as possibly metastatic, more particularly as there was no possible primary tubercular focus except a partially calcified bronchial gland. Microscopic examination demonstrated that the little bodies were pseudo-tubercles and that amongst the giant cells were enclosed little remnants of vegetable food. The ulcer was non-malignant in character.

Dévé (*Revue de Chirurgie,* July, 1902, page 67), in an article entitled "Des Cholérragies Internes," collates thirteen instances of " Cholépéritoine Hydatique." Amongst these thirteen are four in which pseudo-tuberculosis of the peritoneum had occurred. The causal irritants were the hydatid hooklets or small pieces of hydatid membrane. In one case (De Quervains, reported in the *Centrabl. f. Chir.* 1897, No. 1), much difficulty in diagnosis resulted, even though the ascitic fluid was bile-stained. In another case (Debove and Soupauft, *Soc. méd. des. hôp.,* 9th Dec., 1902, *et* 19th October, 1894), an actual error was made and the abdomen closed in consequence. Dévé's description of these tubercles is so clear that I quote him in full. He says:

These pseudo-tubercles are commonly of the size of a pin-head or millet-seed; they are whitish or translucent. Macroscopically they are often indistinguishable from genuine miliary tubercles, and appear as though modelled after these lesions. They may occur over the parietal or visceral peritoneum or in the omenta. They are covered by the peritoneal endothelium and appear as though embedded in the subendothelial peritoneal cellular tissue. By introducing under them the taut finger they are rendered more salient. Microscopically, they may resemble miliary tubercles even down to the finest histological detail. Many multinucleated giant cells are present in the centre of these newly-formed little nodules. The giant cells are surrounded in their turn by a mixed layer of epithelioid and round cells. But characteristic or pathognomonic of these pseudo-tubercles are the causal irritants which are found lying amongst the giant cells. In the cases collated by Dévé pieces of laminated hydatid cyst membrane, or the characteristic hooklets, were thus included. Calcification even may take place in some of the nodules. He draws particular attention to these bodies and notes the absence of all allusion to them in French books, and the liability of the surgeon to diagnostic error. He believes their formation to be a protective process and remarks that they may occur on other serous membranes than the peritoneum. Thus he quotes Lehne who recorded a similar pseudo-tuberculosis of the medullary meninges occasioned by the rupture of an hydatid cyst of the spinal column. He further adds that not all the little bodies found in the cases he records were of the nature described above, some being the early stage of secondary hydatid cyst development.

The non-tubercular origin of these little structures is convincingly established, for tubercle bacilli can neither be demonstrated in section, by culture, or by guinea-pig inoculation; and, moreover, the characteristic lesions can be, and frequently have been, produced experimentally by the injection of tubercle free foreign bodies. Thus, Wallenburg produced such lung pseudotubercles by intra-tracheal injection of droplets of metallic mercury. Dévé produced them in rabbits by the injection of hydatid scolices, etc.

I have not been able to find any record of similar cases in English or American literature, but in Rolleston's "Diseases of the Liver, Etc.," in the discussion of the results of intraperitoneal rupture of liver hydatid cysts, brief mention is made of these pseudo-tubercles, and Dévé's article referred to, whilst in Herman's "Diseases of Women," page 776, the following pregnant paragraph is to be found: "The question has been raised but never settled, as to those cases in which the peritoneum is studded with little bodies looking and feeling like miliary tubercles,—whether these bodies are really the product of the tubercle bacillus? In the cases that recovered there is no evidence for or against."

The following history is of interest:

A. B., a middle-aged plump woman was admitted into the German Hospital under the care of Dr. Jellinek, to whom I owe the privilege of seeing the patient and reporting the case. She was a feckless individual and gave a very unsatisfactory history. We eventually learned that she had been seriously ill for six weeks, and that her illness had begun acutely with pain in the lower abdomen, which she insisted upon ascribing to coition vaginal injury. There was no temperature; the face was pale; the pulse quick and feeble; there was marked aversion to food, but no vomiting, and no obstruction symptoms. Her bowels were reported to have moved the day previous. The lungs were normal. The heart sounds were feeble, but no murmur was audible. Examination of the nervous system was negative. The abdomen was well clothed with fat, was somewhat distended, and over the right ovarian region a linear scar was present. There was no rigidity of the abdominal wall, but some tenderness was evident on slight pressure over the bladder region. The liver and spleen were not palpable. The percussion note was dull, and free fluid was present in the peritoneal cavity. Posteriorly, there was dullness to percussion up along the ascending colon, and a slight filling out of this area when compared with the corresponding region of the opposite side. Vaginal examination revealed some thickening of the left tubo-ovarian structures. Rectal examination was negative. The urine, small in amount, gave a Diazo reaction, but was otherwise normal. The leucocytes numbered 12,000 per c.m., 70 per cent. being of the polynuclear variety.

In view of the acute onset, her clinical aspect, the leucocytosis, and the post colonic dullness, I believed her to be suffering from a sub-acute infective peritonitis with a localized post-colonic collection of pus, due to a diseased tube or appendix, rather than from a tubercular peritonitis, which appealed to us as the most likely alternative diagnosis. She was referred to the surgical side and her abdomen opened. A large quantity of brownish serous fluid was evacuated. The intestines and peritoneum that presented were seen to be covered with innumerable miliary tubercles, though (and we would emphasize the point) no matted fibrous bands or enlarged glands were evident. A diagnosis of tubercular peritonitis was made, the abdomen emptied of fluid as far as possible and then closed. The patient continued to weaken and shortly died. Even after the operation I could not correlate her appearance and the clinical findings with an uncomplicated tubercular peritonitis, and so sent her to the postmortem room with a diagnosis of tubercular peritonitis as discovered at operation, plus a peritoneal infection. At the autopsy, made by Dr. Ophüls, the small miliary tubercles were much in evidence, being present over the general peritoneum. The intestines were somewhat matted together by recent fibrinous adhesions. There were no old fibrous bands and no abdominal glandular enlargement. The appendix was normal and there was no pelvic infection. The post colonic dullness was due to a localized collection of pus. Examination of the stomach showed a large perforated gastric ulcer saddling the lesser curvature and small carcinomatous nodules were present in the liver, which, however, was not enlarged and was well up under the rib margin.

The macroscopic examination thus revealed:

1. Subacute septic peritonitis secondary to a perforation of a gastric ulcer as the immediate cause of death.
2. Small carcinomatous nodules in the liver.
3. Small miliary tubercles in the peritoneum which, from their naked-eye appearance, might be: (1) Miliary tubercles; (2) miliary carcinomata; (3) pseudo-tubercles. No primary tubercular focus was present. They were thus probably either carcinomatous or pseudo-tubercular.

Microscopic examination by Dr. Ophüls, to whom I am much indebted for the report, demonstrated that the gastric ulcer was carcinomatous in nature and that the peritoneal nodules were pseudo-tubercles containing no bacilli, but showing in their interior little food particles, which were surrounded by giant cells, they thus corresponding to the pseudo-tubercles found in Hanau's patient.

The record of this patient is extremely instructive, inasmuch as the history (true an imperfect one) did not suggest any ailment of the stomach. Moreover, all symptoms were referred to the lower abdomen, and the few clinical signs present pointed to a lesion in that vicinity. Looking back, I believe that in the absence of a reliable history my original diagnosis should have read, " Sub-acute septic peritonitis plus a retro-colonic abscess—origin uncertain," thus leaving the further elucidation to the operating surgeon. I had seen, some years previously, a patient in whom a tentative diagnosis of tubercular peritonitis had been made. Sudden collapse took place and death ensued. The autopsy showed a gastric ulcer which had evidently perforated, had been occluded by the omentum and then reperforated, causing the fatal collapse. In that patient there were, similarly, no symptoms or signs referable to the stomach, but then the ulcer was occluded till just a few hours previous to death. In this case, however, the perforation was a large one and there was no indication of any such occlusion.

From this case history and the history of the collated instances of pseudo-tuberculosis, we learn that exploration of the abdomen should be thorough, and that a too hasty operative diagnosis of tubercular peritonitis is to be avoided, more particularly if old fibrous bands or enlarged intra-abdominal glands be absent, otherwise avoidable fatalities may result. If the ascitic fluid be bile-stained, echinococcic pseudo-tuberculosis will be thought of; on the other hand, if fibrinous or pseudo-myxomatous masses are found in the abdominal cavity a ruptured ovarian cyst will be looked for, but though no free-food particles be recognizable as in the reported case, gastric perforation of some little standing cannot be definitely ruled out without an exploration of the upper abdomen. They further serve to once again draw our attention to a fact long recognized by pathologists, viz., that the demonstration of the tubercle bacillus by staining, culture or inoculation, is the only sure proof of its being a causal agent in the production of any lesion, however much that lesion may anatomically resemble genuine tuberculosis.